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Cephalic phase responses, craving and food intake in normal subjects

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Cephalic phase responses (CPRs) are elicited during exposure to food cues. They gear up the body to optimize digestion or they compensate for unwanted changes during a meal. The cue reactivity model of binge eating predicts that CPRs are experienced as craving for food, thereby increasing food intake and playing a role in abnormal eating behaviour.

The present experiment was designed to measure CPRs in normal women and to examine its relationship with craving, food intake and restraint. Results show that normal subjects do react to food exposure with changes in heart rate, heart rate variability (HRV), salivation, blood pressure, skin conductance and gastric activity. These CPRs presumably gear up the body and presumably do not reflect compensatory responses. Significant correlations between restraint and blood pressure, between blood pressure and craving, and between craving and food intake were also found. These results are in line with the cue reactivity model and suggest that research into physiological CPRs and craving in the field of eating disorders is valuable.

Introduction

Cephalic phase responses (CPRs) of the body are elicited by exposure to the sensory properties of food (e.g. sight, smell and taste) as well as by simply the thought of eating. They can be the direct result of sensory stimulation, but conditioned processes also play a role (Mattes, 1997). When a subject has learned that a cue predicts food intake, cephalic phase responses are elicited by exposure to that cue. CPRs are believed to optimize the digestion, absorption and use of ingested nutrients (Mattes, 1997; Rodin, 1985). Anticipation of the ingestion of food lessens the interruption of the homeostatic balance of physiological processes and it increases the amount of food that the organism is able to eat within one meal. For example, when CPRs are prevented in rats by making the timing of a meal unpredictable, the rats eat smaller meals (Woods, 1991). It seems, therefore, that CPRs play an important role in the amount of food someone tolerates. Tolerance is a phenomenon that is usually associated with the intake of drugs, and it refers to the decreasing effects of a drug with repeated administration. When drug administration becomes predictable, organisms learn to anticipate the administration by showing preparatory responses. These preparatory responses at least partly explain drug tolerance (Glautier & Remington, 1995; Siegel, 1983; Woods, 1991).

CPRs can optimize digestion and lessen the impact of food by gearing up the body and getting it ready to optimize the digestion of food. Indeed it was found that exposure to food increases salivation (Epstein *et al.*, 1997; Franchina & Slank, 1988; Hodgeson & Greene, 1980; Nederkoorn *et al.*, 2000; Wooley & Wooley, 1973, 1981), gastric activity (Stern *et al.*, 1989), and insulin release in humans (Teff & Engelman, 1996; Teff *et al.*, 1991; Secchi *et al.*, 1995). Nothing is known about the human temperature during exposure, but the body temperature of rats has been found to increase prior to a meal (Woods & Strubbe, 1994). Preparing the body for the processing of food as well as possible might, however, also mean that anticipatory physiological changes are made which are in the opposite direction. These

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compensatory responses will counterbalance the disturbing effects of food intake. For example, eating increases the blood glucose and in rats it is found that prior to a spontaneous meal the blood glucose begins to drop (Woods & Strubbe, 1994). Nicolaidis and Even (in Woods & Strubbe, 1994) found that the metabolic rate decreases in rats prior to a spontaneous meal, whereas it increases after food intake. In humans, cardiac output, which partly reflects metabolic rate, also decreases after exposure to the sight and smell of food (Andersen *et al*., 1992). These opposite reactions prevent blood glucose and metabolic rate from increasing too much above the desirable state. Woods and Strubbe (1994) suggest that more parameters might change preprandially in reverse direction. Of some parameters, the changes in response to food cues are not clear. For example, in some studies no effect of food exposure on heart rate, blood pressure and electrodermal activity was found (Andersen *et al*., 1992; Overduin & Jansen, 1996; Sjövall *et al*., 1990), but in another study an increase in these parameters was reported (Vögele & Florin, 1997). And although effects of eating have been found on heart rate variability (Lipsitz *et al*., 1993; Kaneko *et al*., 1995), there is no empirical data on the effects of food exposure.

In sum, CPRs are adjustments of the body to a coming meal, elicited by exposure to food or food cues, either by direct sensory stimulation or by conditioned processes. The body gears up to facilitate the digestion of the food, or its anticipatory responses are compensatory to diminish the negative consequences of food intake. The latter (anticipatory compensatory responses) presumably increase the amount of food one can eat. Of some parameters, the preprandial changes have been thoroughly investigated; yet in the case of others there are still gaps in our knowledge.

Knowing that CPRs play a role in the digestion of food and the tolerance for large meals, it might be hypothesised that they also play a role in eating disorders in which large food intake is a key factor. Subjects with the eating disorders bulimia nervosa and binge-eating disorder are characterized (among other things) by recurrent episodes of uncontrollable eating binges during which a large amount of food is eaten (APA, 1994). CPRs are expected to occur when the eating binges can be reliably predicted. Moreover, because of the frequent extremely large food intake of binge eaters, which perturbs the internal state more than a normal meal does, larger CPRs can be expected in binge eaters than in normal subjects. The cue reactivity model states that these CPRs subjectively are experienced as craving (Jansen, 1994, 1998; Wardle, 1990). The CPRs to the cues that predict food intake thus increase the craving for the food and thereby make it more difficult for the subject to abstain from eating. From this model, it follows that CPRs add to the continuation of eating binges.

Indeed, when subjects with and without eating disorders are exposed to food cues, more craving (Bulik *et al*., 1996; Fedoroff *et al*., 1997; Karhunen *et al*., 1997) and physiological reactivity is found in the subjects with abnormal eating patterns. For example, increases in blood pressure and electrodermal activity in binge-eating subjects (Vögele & Florin, 1997) and increased activity in the frown muscle (the corrugator) in restrained eaters (Overduin *et al*., 1996, 1997) were found during food exposure. Some studies found a larger increase in salivation after food exposure in subjects with abnormal eating behaviour as compared to normal subjects (Klajner *et al*., 1981; LeGoff *et al*., 1988; Tepper, 1992; Tuomisto *et al*., 1999). Others found no effects or even a decrease in salivation (Bulik *et al*., 1996; Karhunen *et al*., 1997). Teff and Engelman (1996) report a correlation between dietary restraint and cephalic phase insulin release, whereas other studies report no effects of food exposure on insulin release in subjects with eating binges (Karhunen *et al*., 1997), bulimic women and chronic dieters (Broberg & Bernstein, 1989). No differences were found between normal women and female binge eaters in free fatty acids and plasma glucose as response to food exposure (Karhunen *et al*., 1997).

However, drawing a general conclusion from these findings is problematic. In the studies reported, all kinds of subjects (bulimic patients, obese binge eaters, restrained eaters), diverse cues (standardized food, favourite palatable food items, slides of food) and a variety of physiological parameters were used. In addition, although cue reactivity was repeatedly found, it is not always clear whether the reactivity reflects cephalic phase responses. In most cases, alternative explanations can even not be ruled out. It is possible that in subjects with an eating disorder, exposure to "forbidden" food elicits arousal and emotions, which can add to the CPRs or even overrule those responses (Vögele & Florin, 1997). Moreover, the data reviewed above do not make clear whether an increase in craving for food is related to CPRs, which is postulated by the current cue reactivity model (Jansen, 1998).

Although research into the role of craving and CPRs in eating disorders is interesting and promising, the premises upon which the research is based are still hypothetical. Before further research with patients is undertaken, the fundamentals ought to be examined. First, the appropriateness of some parameters for measuring CPRs is still not clear (i.e. heart rate, blood pressure and heart rate variability), although these parameters are used in cue exposure experiments (e.g. Vögele & Florin, 1997). In case of cue reactivity, we do

not yet know what reactivity in these parameters exactly means. Does it reflect a gearing up of the body or is it a compensation for the food-induced changes? A second point concerns the prediction of the cue reactivity model: are CPRs indeed experienced as craving? Third, the relationship between CPRs and meal sizes is still unclear. Is the magnitude of CPRs indeed related to the amount of food someone can eat? And a final question of importance is whether subjects who sometimes overeat show larger CPRs than normal subjects.

In the present study, normal subjects were exposed to palatable food and after the exposure they were instructed to eat a large amount. During the intense exposure as well as during and after the meal, physiological and subjective reactivity was measured. Relationships between the physiological measurements, subjective states, caloric intake and overeating tendency were examined.

Methods

Subjects

Posters in the university building invited healthy female students to participate in a study on the physiological effects of smelling and eating food. The subjects who volunteered were told that eating a lot of food was part of the experiment. Twenty-four normal weight and non-dieting women were willing to participate. They were instructed not to eat for 5 h before the experiment. According to the paper and pencil compliance check, all subjects complied with the instructions and were included in the data-analysis. The women had a mean body mass index (BMI, weight (kg)/height^2 (m)) of 21·5 (*SD* 2·0), their mean age was 20·1 year (*SD* 1·9) and their mean score on the restraint scale was 9·4 (*SD* 4·1).

Measurement

Subjective states

During the experiment 7 subjective states (hunger, anger, relaxation, fear, sadness, craving for food and nausea) were repeatedly rated by the subject on visual analogue scales ranging from 0 (i.e. not feeling hungry at all) to 100 (feeling very hungry).

Restraint scale

A Dutch translation of the restraint scale (RS; Herman & Polivy, 1980) was used. The RS is a self-report questionnaire consisting of 10 items assessing attitudes towards weight and eating as well as the frequency of dieting and weight fluctuations. Scores range from 0 (no restraint) to 35 (high restraint). Subjects scoring high on the RS, are characterized by an eating pattern alternating between dieting episodes and periodic overeating (Herman & Polivy, 1980).

Physiological measurements

Electrophysiological recordings were all sampled at 250 Hz and in total 24 recordings of 4 min-episodes were made.

Heart rate was measured using two Ag–AgCl electrodes, one attached on the left side of the subject, the other attached under the right collarbone. R-waves were detected off-line with a template matching procedure, and inter-beat intervals were calculated.

For heart rate variability the power was calculated in three frequency bands: respiratory frequency (RSA, 0·15–0·4 Hz), low frequency (LF, 0·05–0·15 Hz) and very low frequency (VLF, 0·003–0·05 Hz) (Bernston *et al*., 1997).

Blood pressure was transduced using a Finapress Monitor (Ohmeda); the Finapress cuff was attached around the middle phalanx of the middle finger of the non-dominant hand. The hand was placed on the table, at a height just below the heart region.

Peripheral pulse amplitude was measured by finger pulse photoplethysmography, placing the sensor on the index finger of the non-dominant hand.

Electrogastrography was measured using three Ag–AgCl electrodes, one placed on the subject's left side approximately 6 cm from the midline and just below the lowest rib, another just above the umbilicus and a reference electrode placed on the subjects left ankle. Periods with artefacts were not analysed. The data were resampled at 25 Hz, mean and linear trends were removed, and were filtered with a high pass filter (at 0·0184 Hz, −3dB, 21) and windowed with a cosine bell window (10%). After this preprocessing, the data segment was Fourier transformed and the power in the frequency band 2·5–3·5 cpm was computed. Because of large differences between subjects, ratio scores with the baselines were calculated.

As a measure of salivation, swallows were recorded with electromyography and the number of swallows was counted. This proved to be a valid and non-invasive method (Nederkoorn *et al*., 1999; Pomerleau *et al*., 1983). The accompanying EMG signal was recorded with three Ag–AgCl electrodes, two electrodes were attached under the left jaw, in the length of the anterior part of the musculus digastricus and a reference electrode was placed on the left mastoid process. Before digitisation, a bandpass filter was set between 10 and 300 Hz, and the signal was rectified and integrated. The digitised epochs were lowpass filtered at 0·4 Hz

(−3dB, lowpass, Ruchkin & Glaser, 1978) to smooth the signal. Artefacts like coughing or talking were removed, and by visual inspection of the data, the definition of a swallowing response was determined as a response of the integrated signal above a 5 μV threshold, with a minimum of 1·5 s between responses.

Skin conductance level was measured with two Ag–AgCl electrodes, placed on the thenar and hypothenar eminences of the palm of the non-dominant hand; respiration rate (cpm) was measured with a strain gauge around the subject's middle.

Temperature was measured by a thermistor, placed on the skin in the proximity of the liver. This is thought to be the most sensitive place for measuring changes in surface temperature caused by metabolic process (Westerterp Plantega *et al.*, 1990).

Because of artefacts during movements, swallows were not measured during the second exposure period and both swallows and respiration rate were not measured during the eating period.

Procedure

At the moment a subject registered for participation, she rated diverse kinds of candy and meals, ranging from 0 (do not like it at all) to 10 (like it very much). The ratings were used to select the favourite kinds of food of the subject. For each individual, the experiment started at 1:00p.m. or 5:00p.m. When the subject came in the laboratory, the electrodes were attached and the subject was instructed to complete the subjective ratings. The subjective ratings were repeated 24 times during the experiment (see the arrows in Fig. 1). There were six measurement periods (see Fig. 1): baseline (8 min), exposure (8 min), intensified exposure (8 min), second baseline (8 min), eating period (32 min) and a third baseline or rest period after eating (32 min). During the baseline measurements the subject was instructed to sit motionless and to relax. During the food exposure, three plates with diverse kinds of preferred food were placed on a table in front of the subject. She was instructed to bend over the food, to look at it and to smell it, and to imagine how it would taste. After 8 min, the intensified exposure started; the subject was instructed to taste the food by licking it and to expectorate, in order to prevent consumption. After the intensified exposure, the plates were removed and a second baseline recording was made to allow physiological responses come to rest. Then the eating period started; the subject received her favourite meal (heated in the microwave), a dessert and various kinds of candy. She was instructed to eat as much as she could without becoming uncomfortable or sick. She also received a drink of her choice (without alcohol or caffeine). The eating period lasted 32 min. Thereafter, the food was removed and the subject sat quietly for another 32 min. Finally, the subject filled in the restraint scale and a questionnaire about compliance with the instructions and use of medications. Her height and weight were measured and she received a financial reward.

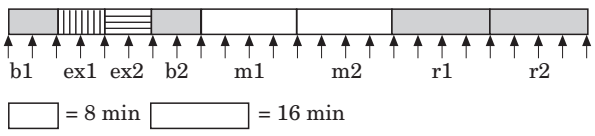

Figure 1. Time scheme of the experiment. ↑, VAS; b1, first baseline; ex1, exposure; ex2, intensified exposure; b2, second baseline; m1, first meal period; m2, second meal period; r1, first rest period after the meal; r2, second rest period after the meal.

Data reduction and analysis

The effects of food exposure and eating were analysed in separate ANOVAs for repeated measures. Greenhouse-Geisser correction was applied when Mauchly's test of sphericity was significant. For the effects of food exposure the average over 8 min was calculated; for the effects of eating an average over 16 min was calculated. If the ANOVA was significant, simple contrasts were specified with the baseline as reference (the first baseline in the exposure analyses, the second baseline in the eating analyses) in *post hoc* analyses. Because of technical failure, blood pressure of five subjects was not measured and because of too many artefacts in the recorded signals, four subjects were removed from the EGG-data analyses and two subjects of the swallow analyses.

Results

Food exposure

The subjective ratings indicated effects of food exposure on craving for food ($F(3, 69) = 16{\cdot}6$, $p < 0{\cdot}001$) and hunger ($F(3, 69) = 10{\cdot}1$, $p < 0{\cdot}001$), but not on anxiety, anger, satiety, sadness, nausea and relaxation. The *post hoc* contrasts showed that craving and hunger increased during the exposure and decreased a little during the second baseline, though were still elevated compared to the first baseline (see Fig. 2).

The overall ANOVAs revealed significant increases of heart rate ($F(3, 69) = 14{\cdot}8$, $p < 0{\cdot}001$), the low frequency component of heart rate variability (LF, $F(3, 69) = 8{\cdot}6$, $p < 0{\cdot}001$), diastolic ($F(3, 54) = 23{\cdot}2$, $p < 0{\cdot}001$) and

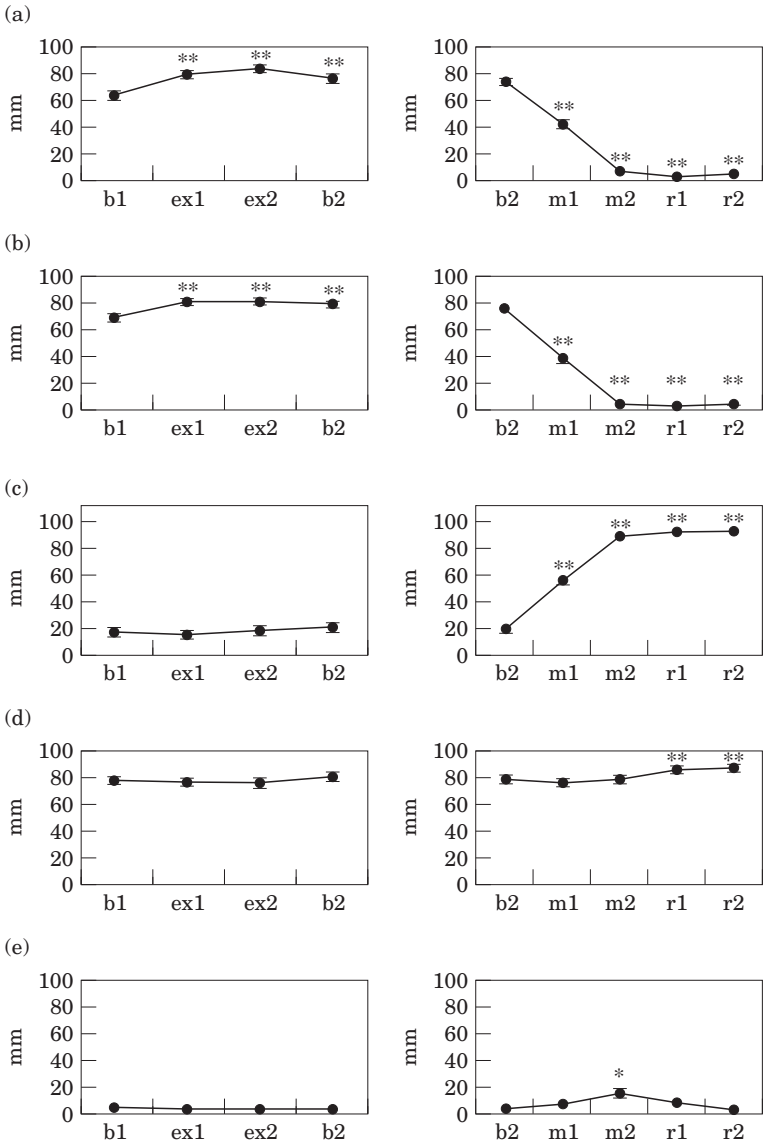

Figure 2. Mean subjective ratings of (a) craving; (b) hunger; (c) satiety; (d) relaxation; (e) nausea, with *SEM* during food exposure and binge eating (large meal). b1, first baseline; ex1, exposure; ex2, intensified exposure; b2, second baseline; m1, first meal period; m2, second meal period; r1, first rest period after the meal; r2, second rest period after the meal. $*p < 0{\cdot}05$, $**p < 0{\cdot}01$.

systolic blood pressure ($F(3, 54) = 15{\cdot}8$, $p < 0{\cdot}001$), temperature ($F(3, 69) = 4$, $p < 0{\cdot}05$), skin conductance level ($F(3, 69) = 19{\cdot}5$, $p < 0{\cdot}001$), and number of swallows ($F(2, 42) = 12{\cdot}2$, $p < 0{\cdot}001$) during food exposure. The RSA component of heart rate variability decreased significantly ($F(3, 69) = 3{\cdot}9$, $p < 0{\cdot}05$). A marginally significant effect was found on gastric activity, which increased during the exposure ($F(3, 57) = 3{\cdot}7, p = 0{\cdot}065$). No effects were found on the very low component of heart rate variability (VLF), finger pulse amplitude and respiration rate. Contrasts showed that heart rate, HRV-LF, skin conductance and number of swallows all significantly increased during food exposure and decreased again to a non-significant level during the second baseline, compared to the first baseline. Diastolic and systolic blood pressure increased during food exposure and remained elevated during the second baseline; temperature remained marginally significantly elevated. A third pattern can be seen in the respiratory sinus arrhythmia of heart rate variability (HRV-RSA); food exposure had no significant influence on this component, but during the second baseline the power in this frequency decreased significantly. All changes are shown in Fig. 3.

Effects of a large meal

The mean caloric value of food eaten by the subjects was 1277 kcal (*SD* 271). There was no significant difference in the amount eaten between subjects who started the experiment at 1:00p.m. and subjects who started at 5:00p.m. ($F(1,23) = 0{\cdot}94$). The large meal had a significant influence on hunger ($F(4,92) = 190{\cdot}9$, $p < 0{\cdot}001$), craving ($F(4,92) = 218{\cdot}6$, $p < 0{\cdot}001$), satiety ($F(4,92) = 154$, $p < 0{\cdot}001$), nausea ($F(4,92) = 8{\cdot}8$, $p < 0{\cdot}005$) and relaxation ($F(4,92) = 11{\cdot}2$, $p < 0{\cdot}005$) (see Fig. 2). Contrasts reveal that hunger and craving for food were high during the baseline, decreased during the meal, and stayed low during the resting period. Ratings of satiety showed the reverse pattern: satiety was low during baseline, increased during the meal, and stayed high during the resting period. Nausea significantly increased during the final eating period, and decreased to a non-significant level during the resting period. During the resting period after the meal, subjects were more relaxed than during the baseline or during the meal. No effects were found on anxiety, anger and sadness.

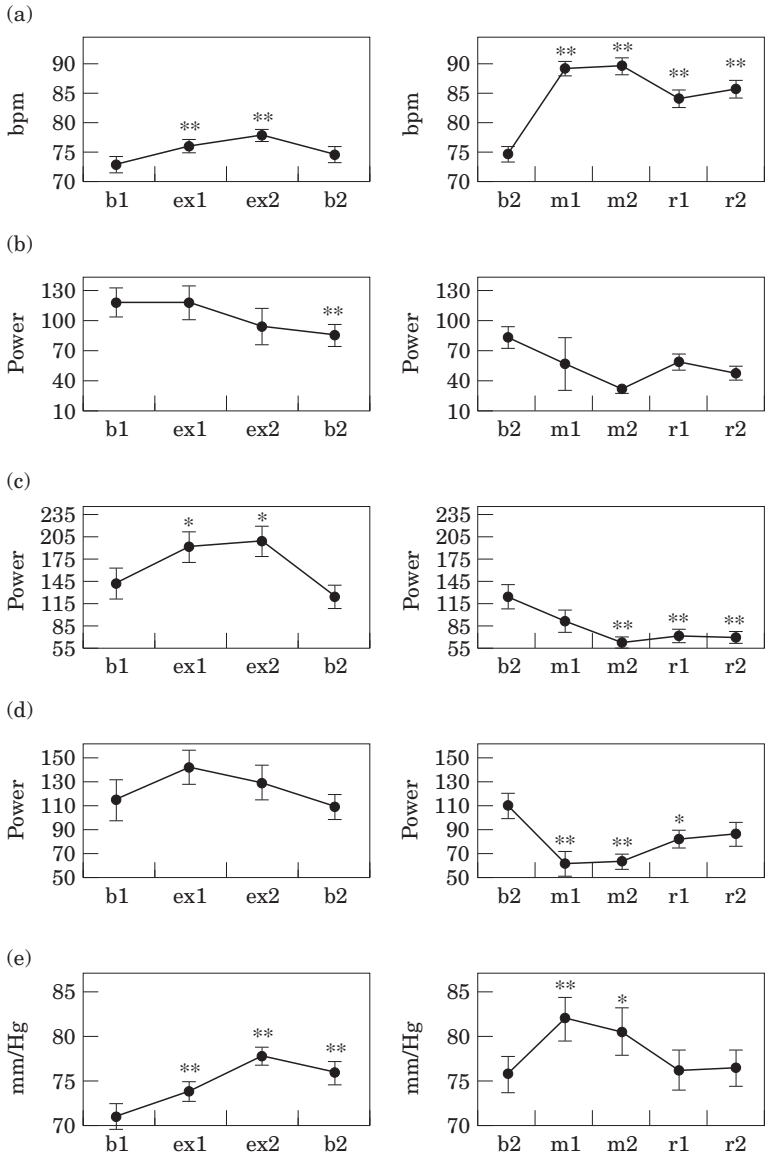

Figure 3. Mean physiological reactions of (a) heart rate; (b) HRV-RSA; (c) HRV-LF; (d) HRV-VLF; (e) DBLP; (f) SBLP; (g) finger pulse amplitude; (h) gastric activity; (i) temperature; (j) SCL and (k) number of swallows, with *SEM* during food exposure and binge eating (large meal). b1, first baseline; ex1, exposure; ex2, intensified exposure; b2, second baseline; m1, first meal period; m2, second meal period; r1, first rest period after the meal; r2, second rest period after the meal. $*p < 0{\cdot}05$, $**p < 0{\cdot}01$.

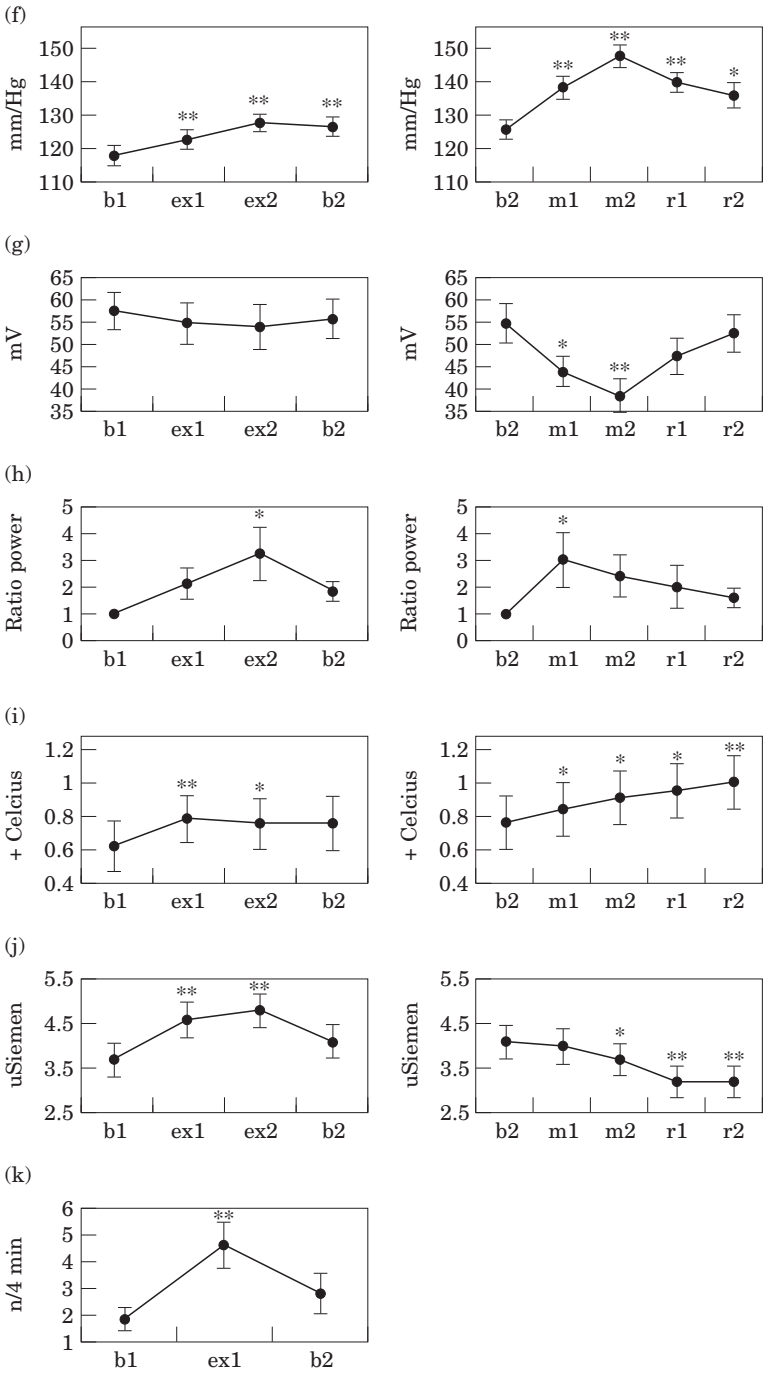

Figure 3 (continued).

There were significant influences of the meal on heart rate ($F(4,92) = 64$, $p < 0{\cdot}001$), heart rate variability (LF ($F(4,92) = 7{\cdot}8$, $p < 0{\cdot}005$) and VLF ($F(4,92) = 6{\cdot}6$, $p < 0{\cdot}005$)), diastolic ($F(4,72) = 7{\cdot}1$, $p < 0{\cdot}005$) and systolic blood pressure ($F(4,72) = 15{\cdot}3$, $p < 0{\cdot}001$), finger pulse amplitude ($F(4,92) = 4{\cdot}9$, $p < 0{\cdot}01$), skin temperature ($F(4,92) = 6{\cdot}0$, $p < 0{\cdot}005$) and skin conductance ($F(4,92) = 15{\cdot}6$, $p < 0{\cdot}001$). A marginally significant increase in gastric activity was found ($F(4,76) = 3{\cdot}0$, $p = 0{\cdot}066$) (see Fig. 3). Contrasts revealed that during and after the meal heart rate was elevated. The power in the low components of heart rate variability decreased significantly during the second eating period and remained low after the meal. The power in the very low components of heart rate variability declined during both eating periods and the first resting period;

during the second rest period after the meal it increased again to a non-significant level. Diastolic blood pressure increased during the meal and decreased again after the meal; systolic blood pressure increased during the meal and stayed elevated after the meal. Finger-pulse amplitude decreased during the meal and increased again to a non-significant level after the meal. This indicates that during the meal there is peripheral vasoconstriction. The skin temperature increased during and after the meal. Skin conductance level decreased during the second meal period and after the meal. Power in the main frequency of gastric activity showed a marginally significant increase during the meal and decreased after the meal. There was no significant effect of the meal on the RSA component of heart rate variability.

Relationship between CPRs, subjective craving and hunger and the intake of food

Partial correlations between CPRs, craving, hunger and food intake were calculated, thereby controlling for the subjective ratings of anger, relaxation, fear, sadness and BMI. First, the relation between cue reactivity on the one hand and craving and hunger on the other was tested. Ratings of craving during the first exposure period correlated significantly with diastolic blood pressure ($r = 0{\cdot}62$, $p < 0{\cdot}05$) and systolic blood pressure ($r = 0{\cdot}64$, $p < 0{\cdot}05$). Hunger did not correlate significantly with CPRs.

To test the hypothesis that subjects with increased reactivity are able to eat more, the amount of calories ingested during the meal was correlated with CPRs. No significant correlation between physiological reactivity and food intake was found. There was, however, a positive correlation between the increase in subjective craving during the first baseline period and the amount of ingested calories ($r = 0{\cdot}58$, $p < 0{\cdot}05$).

Partial correlations between restraint scores, CPRs, craving and meal size were also calculated, thereby controlling for emotions and BMI. Restraint correlated with diastolic blood pressure ($r = 0{\cdot}66$, $p < 0{\cdot}05$) and systolic blood pressure ($r = 0{\cdot}64$, $p < 0{\cdot}05$) during food exposure.

Discussion

The present experiment was designed to study the relationships between: (1) physiological reactivity first during food exposure and then during food intake; (2) craving and CPRs; (3) CPRs and food intake; and (4) restrained eating behaviour and CPRs.

Concerning (1) the physiological reactivity during food exposure and food intake, it can be concluded that food exposure caused a strong increase in hunger and craving. Exposure also had a pronounced effect on physiology: salivation, temperature, heart rate, HRV-LF, HRV-RSA, skin conductance, diastolic and systolic blood pressure all changed significantly during the exposure. The changes in physiological reactivity during food exposure and food intake were in the same direction for saliva, temperature, blood pressure, heart rate and gastric activity. The present findings on saliva and gastric activity are consistent with earlier findings (Epstein *et al.*, 1997; Franchina & Slank, 1988; Hodgeson & Greene, 1980; Nederkoorn *et al.*, 1999; Stern *et al.*, 1989; Wooley & Wooley, 1973, 1981), and the increase in temperature during food exposure is in line with the preprandial rise in temperature found in rats (Woods & Strubbe, 1994). Also an increase in heart rate during the meal has been found earlier (Vaz *et al.*, 1995). A heart rate increase during exposure was found once (Vögele & Florin, 1997), but not always (Andersen *et al.*, 1992; Overduin & Jansen, 1996; Sjövall *et al.*, 1990). Andersen *et al.* (1992) found a decrease in cardiac output during food exposure, whereas we found a heart rate increase. Note, however, that cardiac output is not the same as heart rate. Cardiac output is determined by the strength and frequency of heart contractions as well as the resistance against which the heart is pumping. We did not measure cardiac output, unfortunately.

Opposite changes during exposure and during the meal were found in the low frequency (LF) of heart rate variability and in the skin conductance. The LF variability of heart rate is a product of both sympathetic and parasympathetic influences and is often thought to be an index for mental effort or cognitive processes (Berntson *et al.*, 1997). The increase found during the exposure might reflect the mental effort that is required when concentrating on the smell, sight and taste of the food. Skin conductance, which is an index of activity of sweat glands, increased during food exposure and decreased during and after the meal. Skin conductance can be interpreted as a measure of arousal. The increase during food exposure and the decrease during and after the meal is in accordance with the changes in the LF of heart rate variability. The decrease of the LF of HRV and of skin conductance during and after the meal might be the result of increased familiarity or boredom of the situation, which perhaps decreased arousal and attention. The subjects also reported a significant increase in relaxation after the meal. It does not therefore seem feasible that the changes serve a compensatory role.

The RSA variability of heart rate (HRV) decreased during the second baseline. HRV-RSA is generally believed to provide an index of vagal activity, and is found to be influenced by respiratory rate and depth (Berntson *et al.*, 1997). Why the subjects showed less

vagal activity during the second baseline is not clear. During the meal, no significant changes were found. The very low frequency of heart rate only changed during the meal, as did finger pulse amplitude.

Our findings of an increase in diastolic and systolic blood pressure during food exposure were reported before (Vögele & Florin, 1997), although the documented effects of eating on blood pressure are inconsistent. Both increases in systolic blood pressure (De Mey *et al*., 1989; Vaz *et al*., 1995) and a lack of changes (Lipsitz *et al*., 1993; Kaneko *et al*., 1995) are reported. Probably the large size of the food intake contributed to the increase found during and after the meal in this study.

In conclusion, the CPRs found in this study can mostly be interpreted as gearing up the body to prepare for food intake. The low frequency of HRV and electrodermal activity changed in opposite directions, but an interpretation in terms of compensatory responses is questionable.

Second, the prediction of the cue reactivity model that CPRs are subjectively experienced as craving was tested. Significant correlations were found between craving and systolic and diastolic blood pressure. Although correlations do not indicate a causal relationship and not all CPRs significantly correlated with craving, this finding does support the hypothesis that at least some CPRs are experienced as craving.

The third point under study was the question whether CPRs were related to the amount of food a subject eats. They were not; we did not find a significant correlation between CPRs and food intake. The subjects were pressed to eat as much as possible and it cannot be ruled out that, if the subjects were given the opportunity to eat as much as they liked without pressure, a relationship would be found between CPRs and food intake. The increase in subjective reactivity (craving) during food exposure, however, was significantly correlated to food intake. This finding supports the role of craving in the regulation of food intake.

Finally, with respect to the fourth point whether subjects' restraint is related to the amount of CPRs, it was found that systolic and diastolic blood pressure correlated significantly with restraint scores. Thus, subjects who are inclined to alternate between dieting episodes and periodic overeating showed more physiological reactivity during food exposure. This effect is robust, the more because the absolute restraint scores were quite low and there was not much variability between subjects. Nevertheless, a strong relation between restraint and CPRs was found, suggesting that exposure to food in particular elicits CPRs in restrained eaters. This finding is in line with the cue reactivity model, which holds that a more chaotic eating pattern (alternating between restricted eating and overeating) will induce larger CPRs during food exposure (see Jansen, 1998). Research into CPRs thus might benefit from using highly restrained eaters.

In the present study, we found several interesting connections. A restrained eating style was significantly related to blood pressure, blood pressure was significantly related to craving, and craving was significantly related to food intake. There was, however, neither a direct relationship between restraint on the one hand and craving or food intake on the other, nor a direct relationship between blood pressure and food intake. A common underlying factor might explain the diverse relationships. This factor is not BMI, nor emotionality, because by using partial correlations we controlled for the influences of weight and emotions. What other factor(s) might have influenced blood pressure, craving and food intake? The predictability of eating high calorie food and the quantity eaten will determine the strength or intensity of a learned association between cues and high calorie food. In its turn, the strength of conditioning will influence the size of CPRs, craving and food intake. Then, subjects eating large amounts of high calorie food in a predictable way will show larger CPRs, craving and food intake.

Although the subjects were not explicitly observed in the present experiment, they might have felt observed. Apart from the electrodes on their body, the experimenter entered the lab every 4 min to present a questionnaire. This might have inhibited food intake of, in particular, the more restrained subjects ("eating less in the presence of others and eating more when alone" is one of the items in the restraint scale). Although highly restrained subjects ate less than unrestrained subjects, their physiological reactivity to food cues was stronger. Following this line of reasoning, one would expect that when restrained eaters are alone at home, their stronger physiological reactivity in the presence of food cues would disinhibit them, ending up in increased food intake. In social and laboratory situations, inhibition because of being observed could counteract this effect. Further research is needed to sort out the factors that influence CPRs, craving for food and caloric food intake.

Food exposure leads to physiological reactivity in normal subjects and this reactivity can be measured by non-invasive techniques. The responses found in the present study might be interpreted as gearing up the body to digest the coming food as well as possible. Individual differences in restrained eating correlated to the size of the cephalic phase responses, namely systolic and diastolic blood pressure. Blood pressure also correlated with subjective ratings of craving. Craving for food, in turn, was significantly correlated to food intake.

These results thereby support the cue reactivity theory and confirm that research into CPRs and craving in the field of eating disorders is valuable.

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